PBDE 153 AND THYROID FUNTION IN HEALTHY PREGNANT WOMEN

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Background and Aims: Prenatal exposure to environmentally relevant levels of PBDEs may impair normal brain development by interfering with adequate maternal thyroid function, which is essential to fetal brain development.[1, 2] Our objective was to examine the relationship between prenatal exposure to PBDEs and maternal thyroid function among a cohort of women with normal thyroid function.
Methods: We recruited 314 pregnant women from prenatal clinics in New York City. Eligible subjects were free from clinical thyroid disorders and reported no history of thyroid cancer. We measured concentrations of 12 PBDEs congeners, free thyroxine (FT4) and thyroid stimulating hormone (TSH) in maternal blood samples collected in the first and second trimester of pregnancy (range 5-23 weeks gestation). The association between maternal thyroid function and prenatal exposure to PBDEs was evaluatedusing linear and logistic models controlling for potential confounders including parity, maternal age, education, income and gestational age at thyroid function testing.
Results: The majority of women (81%) demonstrated normal thyroid function, 9% of women had subclinical hypothyroid (elevated TSH, normal FT4), 7% hypothyroxinemia (normal TSH) and 3% overt thyroid disease. All 12 PBDE congeners were detected in maternal plasma (detection frequencies ranged from 6.7% to 98.4%). Congeners 47, 100, 99 and 153 were detected with the greatest frequencies (99%, 91%, 83% and 98.4%, respectively). PBDE 47, 99 and 100 were highly correlated with each other ($r > 0.8$, $p < 0.05$). PBDE 153 was not correlated with any other congeners. Though not statistically significant, PBDE 153 was inversely associated with the FT4 levels dichotomized at the lowest 5%, OR (95%) CI 1.44 (0.89 to 2.32) $p = 0.13$ after adjustment for lipids, triglycerides, parity and gestational age at thyroid function testing. No other PBDE congeners were associated with maternal FT4 or TSH levels.
Conclusions: Our results suggest prenatal exposure to PBDE 153 may be inversely associated with circulating FT4.
References: Chevrier J, Harley KG, Bradman A, Gharbi M, Sjodin A, Eskenazi B. Polybrominated diphenyl ether (PBDE) flame retardants and thyroid hormone during pregnancy. *Environ Health Perspect*. 2010;118(10):1444-1449.

Pop VJ, Kuijpens JL, van Baar AL, Verkerk G, van Son MM, de Vijlder JJ, et al. Low maternal free thyroxine concentrations during early pregnancy are associated with impaired psychomotor development in infancy. *Clin Endocrinol (Oxf)*. 1999;50(2):149-155.